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MYOSITIS OSSIFICANS.

WITH A REPORT OF TWO CASES.[1]

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The comparative rarity of myositis ossificans, its peculiar clinical and anatomical manifestations, and, more than all, the mystery enshrouding its etiology, make the disease one of great interest to the pathologist, the clinician, and the surgeon; and the report of two similar cases, while it may add little to the existent knowledge on the subject and do little to enlighten us as to the etiology of the disease, cannot fail to be of value. As Roth says: "If we no longer, in the present state of our knowledge, consider myositis ossificans as a pathological curiosity, yet, in view of the still small number of published cases and the darkness which still hovers over the whole character of the affection, the demonstration of a patient suffering from this mysterious disease must be interesting."

The autopsies in my cases were made by Dr. Warthin, and it is to his kindness that I am indebted for the post-mortem notes, the material, and many valuable suggestions.

The first of the cases is of a young farmer, aged twenty-three years, the cause of whose death was pulmonary and laryngeal tuberculosis. He died April 11, 1897. His clinical history presents nothing of interest in this connection until March 1, 1897, when he complained of pain in the left leg. On examination the left leg and foot were found swollen, soft, and œdematous. The thigh was also swollen, though less than the leg. On March 28, 1897, examination showed the left leg only slightly œdematous, and it was no longer painful. A marasmic thrombus of the left saphenous vein was believed to have caused the œdema.

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[1] Read before the Washtenaw County Medical Society, March 5, 1900.

The autopsy showed a very general tuberculous process. Both lungs were infiltrated with tubercles and contained large cavities. Small tubercles were found in the spleen, liver, adrenals, and kidneys, and a tuberculous ulcer in the lower part of the ileum. The bronchial glands contained many areas of caseation, the mesenteric glands were enlarged and some of them caseated, and all the lymph glands of the body were enlarged.

In the left saphenous vein was found an old, partially organized thrombus, extending downward for a distance of about ten inches. This extended upward into the femoral vein, which was partially closed by a mixed thrombus, apparently fresh. From the femoral vein the partially obturating thrombus extended into the left external iliac, and through this in constantly decreasing size into the left common iliac and the abdominal vena cava. Around the wall of the femoral vein there was an area of hyperæmia and infiltration, extending apparently from an enlarged lymph gland near by. This gland on section showed infiltration and beginning caseation.

The femoral vessels with the thrombus and the circumjacent fat, connective tissue, and muscle were removed, hardened in Müller's fluid, dehydrated, embedded in celloidin, and sectioned. The sections were then stained in hæmatoxylin and eosin. Some of the sections were counterstained in Van Gieson's picric acid and acid-fuchsin mixture, and others were stained by Unna's orceïn method for yellow elastic tissue.

The microscopical appearance is as follows: Both femoral vessels are nearly or quite filled by thrombi in various stages of organization.

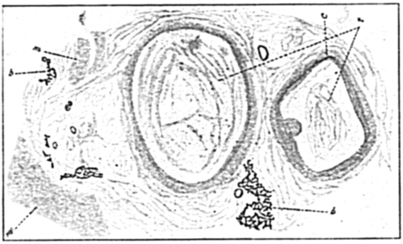
The whole section shows marked hyperæmia, all the smaller vessels and even the capillaries being distended and packed full of blood corpuscles. The connective tissue surrounding the large vessels, as well as that around the smaller ones, is infiltrated with leucocytes. Especially around the distended capillaries are seen aggregations of small round cells. This leucocytic infiltration is especially marked in the intermuscular connective tissue and around the capillaries found in that connective tissue. Many of the leucocytes are markedly degenerated, and in some parts of the sections larger or smaller necrotic, abscess-like areas are found. Surrounding these necrotic areas, and indeed in many parts of all the sections, attempts at repair are noticeable. The inflammatory tissue seems to have been replaced by a new granulation tissue, rich in small, thin-walled bloodvessels and in large plate-like cells, with little fibrous tissue.

The most marked changes to be noted are, however, in the muscular tissue. As before stated, the intermuscular connective tissue is hyperæmic and infiltrated with leucocytes. In some parts of the sections, especially at the periphery, the muscle appears fairly normal, but even here, in most of the fibres, the cross striation has disappeared or is less distinct than in normal muscle. The longitudinally cut muscle fibres, which are of normal size near the periphery, become smaller as they near the centre, and may taper to a point or divide into several muscle fibres or seem to resolve themselves into numerous bundles of fibrous tissue. Nearer the centre of the section the muscle fibres are all much smaller than normal, of very irregular contour, with no cross striations, and in some even the longitudinal striation seems lost, the fibres appearing either finely granular or quite homogeneous. In some places a muscle nucleus with a small spindle-shaped bit of protoplasm is separated off from the rest of the cell. The nuclei may be absent from a considerable part of the fibre and crowded together at one end or at one side, and often a small, narrow fragment of muscle containing many nuclei is seen. Some of the longitudinally cut muscle fibres, after decreasing in size, suddenly enlarge, club-like, at the extremity, this enlargement containing large numbers of muscle nuclei. Often small, isolated fragments of muscle are seen in the granulation tissue, and even in and near the inflammatory, necrotic areas, containing numerous nuclei, embedded in homogeneous appearing protoplasm, producing the appearance of giant-cells. We find everywhere marked variations in the size and shape of the muscle fibres, with the ends pointed, branched, or club-like. Along the course of the fibre the nuclei are generally greatly increased in number and are scattered irregularly through the apparently undifferentiated sarcoplasm. Beside the simple atrophy, some of the fibres seem to be undergoing fatty degeneration and some Zenker's waxy necrosis.

In the granulation tissue, which seems to have replaced the larger inflammatory areas, we find irregularly branching and anastomosing trabeculæ of osteoid tissue, consisting of a dense matrix, enclosing rather large cells, which are usually not surrounded by a capsule. These trabeculæ surround smaller areas of granulation tissue which is richly supplied with bloodvessels and which here somewhat resembles bone-marrow. Many of the large cells of these spaces are arranged in a more or less continuous layer on the trabeculæ, like the layer of osteoblasts on the trabeculæ of developing bone. Most of the osteoid tissue has undergone calcification, at least in the central part, so that, according to Ziegler's definition, we may believe that we have here a true ossification process. In some of the sections these plates of bone form a nearly continuous ring around the larger bloodvessels. In others they are scattered throughout the section in smaller masses, always found in the new granulation tissue, never in the old connective tissue, nor in the inflammatory tissue which is still undergoing retrograde changes.

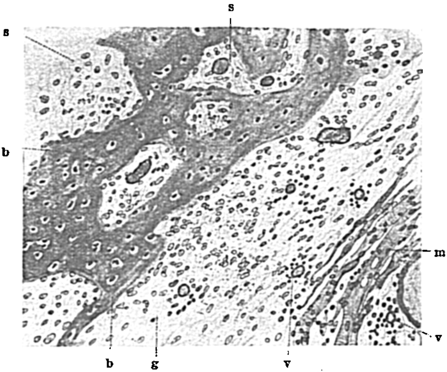
These ossified masses are found not only immediately around the femoral vessels, but extending out in all directions between the degenerating and regenerating muscle fibres, wherever a sufficiently large area of granulation tissue is found. The relation of the plates of bone

FIG. 1.



Section through both thrombosed femoral vessels, muscles, fat, connective tissue and granulation tissue containing plates of bone. m, muscle; b, bone; c, calcification; t, thrombus. Drawn under the dissecting microscope, with aid of camera lucida. Magnified about eight times. Reduced to one-half size of drawing.

FIG. 2.



Section through a few trabeculæ of pathological bone formation, with marrow spaces, surrounding granulation tissue, and, at the periphery, degenerating muscle fibres. b, bone; m, degenerating muscle; g, granulation tissue; s, marrow spaces; v, capillaries surrounded by connective tissue infiltrated with leucocytes. Drawn with the aid of the camera lucida. No. 2 eye-piece; ⅙ in. objective. Reduced to one-fourth.

to the other tissues of the section is represented in Fig. 1. In the marrow spaces we see frequently small bits of degenerated muscle, atrophied, non-nucleated, and appearing homogeneous.

It may not be uninteresting to speak here briefly of the changes in the yellow elastic tissue as brought out by Unna's orceïn differential stain. In the necrotic areas, in the degenerating inflammatory masses, the yellow elastic tissue seems to have disappeared or to have lost its power of taking the differential stain. In some parts of the granulation tissue, on the other hand, we begin to see short pieces of fine fibres, which are well stained with the orceïn, the surrounding tissue remaining unstained, while in the older parts, at the periphery of the larger bone-containing masses the fibres are coarser and more massed together. Even in the bone tissue itself, fine, yellow-elastic fibres are occasionally seen. It seems unquestionable that in this process the yellow elastic tissue, as well as the muscle and fibrous connective tissue, degenerates and regenerates.

The enlarged lymph glands in the neighborhood of this hyperæmic process were removed and examined. No tubercle bacilli were found, nor was the histological structure that of a tubercle, but rather of a simple, necrotic abscess.

The changes in the tissue would seem to indicate that the intermuscular inflammation, the changes in the muscle, and the bone formation antedated by a considerable time the thrombosis, which was probably secondary to the other changes, and was the cause of the clinical symptoms noted about a month before death.

In the second case I have found nothing of interest in the clinical history except the fact that the thigh was broken about five weeks before the man's death.

In this case also there was an obturating thrombus of the femoral vein, and the microscopical appearance is identical with that in Case I. The only difference in the two cases is that in Case I. the process seemed to result from a chronic inflammation (possibly tuberculous), while in Case II. it resulted from an injury to bone, which may have injured also the muscle and other soft parts. It should be stated, however, that the fracture was not near the point of ossification of the muscle.

In both cases we have thrombosis of the femoral vein, which has not occurred in any of the other cases cited in the literature to which I have had access; also periarteritis, periphlebitis, inflammatory infiltration of the intermuscular connective tissue, with degenerative changes in the muscle, and ossification of the granulation tissue.

In both cases, also, the process was not discovered until the autopsy had been made and the sections examined microscopically.

Cases of pathological ossification naturally divide themselves into two great classes: those in which bone is formed in connection with bone, and apparently resulting from an abnormal activity of the cells of the periosteum, and those in which bone is formed in the softer tissues and has no connection, primarily at least, with the skeletal bones or their periosteum. Of the first class I need say nothing further, as the consideration of these cases is quite foreign to my subject. In the second group of cases, myositis ossificans may be said to occupy a very prominent position. Cahen divides this disease into two classes: a progressive form and a localized or stationary form. The progressive type, known as myositis ossificans progressiva, is, according to Cahen, distinguished from the localized form by the facts that it begins in youth, advances periodically, and attacks many series of muscle groups. To this type belong most of the cases recorded in the literature, which is perhaps due to the easier recognition of this type of the disease.

Roth has recently given an excellent review of much of the literature on this subject, and I may be permitted to draw somewhat freely from his review, especially with regard to those works to which I have not had access. The earliest case reported was in 1740, by Freke, in the *Philosophical Transactions*, the description of the case corresponding very exactly with the descriptions now given. In the same journal, in 1741, Copping thus describes a case: "Das ganze Rückgrat war ein zusammenhängender Knochen, von dem sich ein scharfer Rand erhob, der wie eine Handhabe aussah, woran man das Skelet halten konnte. Es waren ihm jedes Jahr aus den Fersen Hörner gewachsen, wie die Sporen bei den Hähnen und die Ueberwüchse von Knochen waren in so grosser Menge da, dass es eben so schwer sein würde, sie zu zählen als die Stalactiten in der Grotto der Calypso." Other cases were noted by Henry (1759), Rogers (1832), Testelin and Danpressi (1844), Hawkins, Wilkinson, and Abernethy.

In 1869 Münchmeyer, giving to the disease the name suggested by v. Dusch, myositis ossificans progressiva, described it so exactly that later writers have been able to add little to his description. He declared the disease to be a constitutional affection of slow course, with periodical exacerbations. The first disturbance in muscular tissue begins with a marked infiltration of the intermuscular connective tissue. The second state—that of connective tissue induration—consists of an excessive growth of intermuscular connective tissue with destruction of fibrillar muscle substance in consequence of pressure atrophy or fatty degeneration. The third state is that of ossification, which begins in the centre of the affected muscle, in the connective tissue ground substance. The disease begins in youth, with local swelling and later loss of function and ossification. Sometimes local and radiating pain is associated with it, and also slight febrile reaction. The skin may be of a higher temperature than normal, and the connective tissue œdematous. The swelling may disappear after two to four weeks, leaving the muscle hard and ossified, a condition which is sometimes temporary, but generally spreads to the origin and insertion of the muscle, and remains permanent. The disease generally begins in the neck and back muscles, spreading and producing ever-increasing deformity. Münchmeyer reports twelve cases, of which nine were in males, which predisposition of males has been noted by other writers, Roth saying that of the thirty-nine cases which he collected from the literature thirty were males.

Some years later Helferich noted the occurrence of a curious malformation in connection with this disease—microdactylie, an anchylosis of the phalanges of the thumb and a lack of one phalanx of the great toe on both sides. Since his time the same curious anomaly has been noted in 75 per cent. of all the cases recorded. In 1869 Münchmeyer collected from the literature twelve cases of this disease. In 1884 Pinter carefully reviewed the literature and reports twenty-two cases with those of Münchmeyer. In 1896 Pincus very exhaustively reviewed the literature of myositis ossificans progressiva, carefully tabulating the thirty-eight cases which he found. Roth, in 1898, in his review of the literature, mentions having found forty cases, including his own, which number, however, includes the four cases of localized myositis reported by Cahen and one by Schwarz. Eleven of his cases were included in the table given by Pincus, leaving twenty-four which, with the thirty-eight of Pincus give sixty-two cases, which were reported by Skinner, Minkiewicz, Zollinger, Billroth, Byers, Florschütz and Gerber, Ditmeyer, Huth, Nicoladoni, Helferich, Partsch, Kümmel, Uhde and Pinter, Krause, Kohts, Sympson, Willett, Godlee, v. Volkmann, Lendon, Bókai, Macdonald, Svensson, Pollard, Brennsohn, Rabek, Virchow, Carter, Paget, Pincus, Mays, Berger, Storham, Maunz and Lexer in addition to those already mentioned. In an addendum Pincus cites cases from Gibney, Kissel, Linsmayer, Lexer, and Rosenstein.

In a careful review of the literature since 1890 I have found, in addition to those cited by Pincus and Roth, cases of myositis ossificans progressiva reported by Boks, Jones, Nissim, Stonham, Bernachi, Martinez, De la Camp, Lockwood, Bollinger, Jacoby and Morian-Essen, making eleven cases, which, with the sixty-seven collected by Pincus and Roth, make seventy-eight cases. Most of the cases to the reports of which I have had access follow so closely the description of Munchmeyer, already given, that a further reference seems unnecessary; but in one case, reported by Kissel, while the clinical picture was typical of the disease, the microscopical examination of one of the hard swellings revealed the presence of no true osseous tissue, but only of young connective tissue with some remnants of altered muscle. In this case also some of the tumors disappeared, leaving no trace, while others broke down, and a puriform liquid was discharged. This case improved somewhat under treatment, and was regarded by Kissel as an incipient stage of the disease. The usual history of the disease is a slow progress with longer or shorter pauses, the general health remaining good, death finally ensuing either from some intercurrent affection or from starvation after the disease has involved the muscles of mastication and deglutition.

Cases of ossification confined to single muscles or groups of muscles have been reported by Podrazki, Schwarz, Cahen, Schmit, Lehman, Sieur, Lunden, Albertin, Hutchinson, Lalesque, Eineri, Ramonet, Gazni, Cordillot, Orion, Tricot, Mouchet and Munro, making twenty-three cases with the two reported by myself. In addition to these, Cahen has collected from the literature previous to 1890 twelve similar cases, which were reported by Volkmann, Virchow, Rokitansky, Schuh, Billroth, Otto, Pitha, Rasmussen, and Meinhold. Some of these cases followed a single severe injury, as in the four cases described by Cahen, in which the growth resulted from the kick of a horse. Other cases resulted from repeated injuries or from continued use or strain, under which heading we may include the ossification of the deltoid and arm muscles in soldiers and of the thigh muscles in riders. This perhaps includes far the largest number of this class of cases. Then we have ossification occurring in the course of a chronic inflammatory process, which may be rheumatic, syphilitic, or tuberculous.

Mays, Cahen, Lexer, Kissel and Kümmel have made microscopical examinations of cases cited by them, and the findings of Cahen and Lexer may be compared with my own; Cahen's, because his cases represent a type similar to mine; Lexer's, because he has examined some of the growths from a case of true progressive myositis ossificans.

In the case described by Cahen the tumor was removed, and after a short time returned and was again removed, this time with the periosteum, to which it had become adherent. The sections from these two growths present somewhat different appearances. At the periphery of the section of the first tumor, and crowded together by connective tissue containing many bloodvessels and large spindle-cells, he finds muscle which has undergone many degenerative changes—hyaline degeneration, increase of nuclei of sarcolemma, loss of striation, etc. In the centre of the tumor he finds irregularly arranged trabeculæ of bone, with an epithelium-like lining of osteoblasts, with many lacunæ and giant-cells, and the spaces filled with a marrow extraordinarily rich in bloodvessels and connective-tissue cells. All through the preparation are scattered bits of muscle, the relation being so intimate that single muscle fibres are completely embedded in bone. In the sections from the second tumor no muscle fibres are seen. There are three distinct zones: a zone of greatly increased connective tissue cells, then a zone of small-celled hyaline cartilage, which, by ingrowth of bloodvessels and giant-cells, is changed into bone. There is no distinct boundary between the cartilage and bone, the cells becoming smaller and losing their capsules, while the intercellular substance becomes denser, more opaque, and stains bluish-red in the hæmatoxylin-eosin double-stain. The development of bone in all cases investigated stood in direct relation with an increase of the intermuscular connective tissue.

Lexer has described quite minutely the microscopical appearance of sections taken from several tumors removed from two cases of myositis ossificans progressiva. Near the periphery of the preparations he finds quite normal muscle fibres. Nearer the centre the muscle fibres are more or less degenerated, the cross striation lost, the muscle nuclei increased, in many cases resembling giant-cells, and the fibre broken up, while the intermuscular connective tissue is increased and infiltrated with leucocytes, especially in the region of the capillaries. The connective tissue contains many cells of different forms arising from division of connective-tissue cells, which may be considered as fibroblasts, while in some places are seen cartilage cells with formation of hyaline cartilage. Nearer the centre the ground substance becomes denser, the former connective-tissue cells lie in small angular spaces and the formative cells lie in rather regular rows on the dense tissue which comprises the osteoid trabeculæ. Later these become calcified, and thus bone is formed. The noteworthy point about this description is its similarity to that given by Cahen and the similarity of both to my own description. This, it seems to me, would indicate that, though the clinical picture differs much, because of the wide-spread distribution of the lesions in the progressive type, yet the pathological changes are nearly the same in the two types of the disease. This may, however, need further evidence.

Much difference of opinion has arisen as to the true character of the bone formation. Virchow places the disease on the border line between inflammation and new growth, and is supported by Lexer, Bollinger and many others.

Mays asserts that it is a true tumor, and is supported by Kümmel, Pinter, Helferich, Pincus, Partsch, Schwarz, Cahen and others. Cahen bases his assertion on the microscopical appearance, especially the fact that new connective tissue, cartilage, osteoid tissue, and bone are found in the same section, thus showing the characteristics of an atypical growth. They consider the inflammatory phenomena and muscle degeneration to be secondary to the tumor formation. Pincus, after a most exhaustive and careful study of the literature of myositis ossificans progressiva and of his own cases, arrives at the following conclusions: The so-called myositis ossificans progressiva multiplex is not a disease, but undoubtedly a tumor belonging to the multiple osteomata and exostoses of Virchow. The process begins in the periosteum, the muscle degenerating secondarily. At the foundation lies an inborn — not hereditary—constitutional anomaly, the Cohnheim theory of embryonic nuclear *anlagen* not sufficing for explanation ; the constitutional anomaly is to be defined as vulnerability (excessive productivity) of the periosteum and connective tissue of the locomotor apparatus. The disease needs for its development an external cause. As etiological factors we have, above all, trauma ; in a less degree rheumatism. Syphilis, rhachitis, myopathy, and trophoneurosis are excluded. The apparently spontaneous cases arising in early life are really of traumatic origin and due to intrapartum injuries.

Nicoladoni advances the hypothesis that the process is a tropho-neurosis comparable to progressive muscular atrophy and pseudohypertrophy of muscle, but finds no active supporters, although Klemm advances some arguments which seem to support his view, while Schwarz, Eichhorst, and Klemm consider it secondary to a disease of the spinal cord.

Since electrical tests, wherever applied, have seemed to indicate an entire absence of any neuropathic factor, except in the cases of Kümmel and Pincus, where the faradic excitability was much reduced in the degenerated mucles, it seems feasible to consider the disease purely myopathic, either primarily inflammatory in character or having the primary characteristics of a new growth. It seems probable that in the progressive form of the disease many of the swellings are true neoplasms, while others, as would seem to be indicated by Lexer's description, result from an inflammatory process.

Nicoladoni says that in consequence of a diffuse interstitial myositis an indifferent tissue arises which can develop in two different ways: scar tissue and bone. Lexer corroborates this from his microscopical findings, as the tissue arising from proliferation of the connective-tissue cells can, by formation of new fibrillæ, develop into a firm, scar-like tissue as well as undergo a further change into cartilage and bone. Dūms believes it the result of irritation of connective tissue through trauma or blood extravasation, and formation of indifferent granulation tissue from which the subsequent developmental changes arise.

In the myositis ossificans limited to single muscles, my observations would, it seems to me, indicate that the primary condition is an inflammatory process in the intermuscular connective tissue, resulting in degeneration of the muscle fibres, either from simple pressure atrophy or from fatty degeneration, so that the name myositis interstitialis ossificans, as suggested by Stonham, seems not inappropriate. The second condition seems to be the establishment of the processes of repair, the formation of a tissue rich in fibroblasts and small bloodvessels, in which white fibrous and yellow elastic tissue fibres are sparingly developed. Before this process proceeds to the formation of mature connective tissue, however, it is interrupted by the change of a part of the tissue to trabeculæ of firm osteoid substance which becomes calcified, while the parts enclosed by the trabeculæ change to bone-marrow. This change is not so anomalous that it need excite great surprise, since, as is well known, the different members of the connective-tissue groups change with especial readiness to some other form of connective tissue. Bone when broken heals by formation of fibrous tissue, which normally changes to bone, but may remain fibrous in adult life, the cartilages of the larynx and trachea ossify, and other similar changes may occur under certain circumstances.

Many factors have been assigned as exciting causes of this affection: such as cold, unsanitary surroundings, trauma, either slight or severe, either a single injury or many successive injuries, irritation, as in the exercise of bone, or chronic disease processes.

This might be satisfactory on the subject of etiology, were it not that the exciting cause, which most authors admit as a necessary factor in producing the disease, is often so slight and trivial that we cannot believe it sufficient to produce the disease in a normal individual. This is especially true in cases of the progressive type, while in the stationary form of the disease the irritant cause which has operated on hundreds of cases has produced ossification in very few. The explanation of these facts has opened a large field for conjecture and speculation. Virchow believes that there is in these individuals an ossifying predisposition, either hereditary or congenital, which he calls "diathesis ossificata sive ossea."

Mays, Kümmel, Pinter, Helferich, Schwarz, Pincus, Stonham, Paget and many others ascribe it to a congenital, not hereditary, predisposition, which they do not define more exactly.

Maunz suggests as a predisposing causative factor a disturbance of embryonic development. He says: "In the *anlagen* of musculature, where in later life pathological bone formations occur, osteoblastic nuclei enter. These nuclei remain dormant so long as they are held in check by the physiological resistance of neighboring tissues. If, however, this be weakened, the bone *anlagen* develop into the pathological bone formation." He explains in a similar way the occurrence of exostoses and osteophytes which are so frequently found in conjunction with the muscular ossification. Those who favor the view of embryonic disturbance cite the frequent occurrence of microdactylie in this disease as an evidence favoring their hypothesis.

Brennsohn and others suggest the idea that the disease is due to atavistic influence, without, however, expressing any positive belief in the hypothesis. The microdactylie is cited as favoring this theory also. Dr. Warthin is inclined to favor this suggestion in the case of the localized form of the disease. He believes that a sharp distinction should be drawn between the two types, the growths being true neoplasms in the progressive form of the disease, while in the localized form, in which he includes the inflammatory ossification and the exercise and riders' bones, whose site is quite distinctly limited to the thigh and shoulder muscles, we have an atavistic reversion to the splint bone of lower animals.

Direct heredity seems, from the history of the cases reported, to play no rôle in the causation of the disease, if we except the fact noted by Paget that in many cases rheumatism and cancer occur in the family history.

Atavistic influence seems to me to be contraindicated by the fact that in the hand and foot, where splint bones are most common in the lower animals, these bony growths rarely occur. In the progressive muscular ossification, beginning in early life, it may, it seems to me, be necessary to admit the hypothesis of a congenital condition consisting of an abnormal activity of osteoblastic elements, or, if we accept the Weisman theory of embryonic development, the persistence of indifferent, undifferentiated mesenchymal cells, which, under the requisite conditions of nutrition, develop abnormally into nodules of fibrous connective tissue, cartilage, and bone, sometimes in connection with the skeletal bones, sometimes in fascia, tendons, ligaments, or intermuscular connective tissue. In the localized form of the disease, however, no such hypothesis seems to me to be necessary. We have here a formation of granulation tissue, a new connective tissue, whose cells may therefore revert to the undifferentiated, indifferent embryonic cell type, mesenchymal cells, which may develop into fibrous tissue, cartilage, or bone, according to the prevalent nutritive conditions. Although Gegenbaur suggests the possibility that specialized osteoblastic nuclei are necessary for bone development, the ordinary connective-tissue cells being incapable of such development, there seems, in these cases, no valid reason for the belief that the cells, which in certain regions act as osteoblasts, are histogenetically different from those which, in other localities, develop into fibrous connective tissue.

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SARCOMA OF THE SMALL INTESTINE.

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During the past two years there have been observed at the Mount Sinai Hospital five cases of intestinal sarcoma, four of which came to autopsy. In three of these cases the clinical picture closely resembled that of appendicitis. In attempting to ascertain whether such a resemblance had before been noted, I found that not only was the clinical picture unknown, but also that there had been no complete review of the subject of sarcoma of the intestine since Baltzer wrote his article in 1893.

In the following paper I shall give the details of the four cases of ours which came to autopsy, and refer occasionally to the fifth case. I shall then attempt to give a complete picture of the disease from all its stand-points. I am indebted to Dr. Gerster for his kindness in permitting me to observe four of these cases clinically, to Dr. Lilienthal, in whose service the fourth case occurred, and to Dr. Brewer, by whom it was operated upon.

Case I.—H. G., aged twelve years, schoolboy, admitted September 30, 1898. History is of nine days' duration. The boy first complained of pain in the abdomen, below and to the right of the umbilicus, and of weakness. Three days ago abdominal distention was noticed, and since then there is marked constipation. The distention and pain have increased, and the boy suffers from dyspnœa.

On admission the temperature was 100.6°. The status in short was the following: Superficial abdominal and thoracic veins distended. Liver: upper border of dulness at fifth space, lower border undetermined. Abdomen much distended, signs of free fluid. Across the hypogastric region and extending into the pelvis there is a nodular tumor, concave above. Rectal examination revealed a large bulging mass anteriorly.

Operation, on October 1st, by Dr. Van Arsdale. The peritoneum was found lined with white nodular masses, and a large amount of chyliform fluid escaped. After the operation the boy gradually became weaker, there developed a left-sided empyema, purulent peritonitis, and otitis media. He died on October 29th.

Post-mortem Examination. (Abstract.) Lungs: large purulent exudate in left pleura. Small abscess in base of left lung. Infiltration of the diaphragm and base of the left lung by new growth. Evidences of healed tuberculosis in the right lung. Spleen: infiltrations on surface.